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ELSEVIER SCIENCE FULL-TEXT ARTICLE

Transforming growth factor-alpha (TGF-alpha) and epidermal growth factor-receptor (EGF-R) immunoreactivity in normal and pathologic brain.

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Transforming growth factor alpha (TGF-alpha) and epidermal growth factor-receptor (EGF-R) immunoreactivity is observed in the majority of neurons, and in maturing astrocytes, in the developing and adult brain of humans and different species of animals. TGF-alpha and EGF-R co-localize in most neurons and maturing astrocytes, suggesting that most TGF-alpha-producing cells are EGF-R-expressing cells. TGF-alpha and EGF-R immunoreactivity decrease in damaged areas following different insults. However, EGF-R appears in reactive glia, mostly reactive astrocytes, within and surrounding the damaged areas. TGF-alpha and EGF-R immunoreactivity is found in neurons of patients affected by Alzheimer's disease and other forms of dementia, and in neurons of patients suffering from epilepsy owing to different causes, thus pointing to the conclusion that TGF-alpha does not play a significant role in these pathologies. However, EGF-R immunoreactivity occurs in reactive astrocytes and microglia in subacute but not chronic lesions in human cases. Since TGF-alpha is a membrane-anchored growth factor, which may be cleaved leading to the formation of soluble forms, and both the membrane-anchored and soluble forms have the capacity to activate the EGF-R, it is feasible that TGF-alpha in the nervous system may act upon EGF-R-containing neurons through different mechanisms. In addition to distant effects resulting from the release of soluble TGF-alpha, local effects may be produced by establishing direct cell-to-cell contacts (juxtacrine stimulation), or in cells expressing both TGF-alpha and EGF-R (autocrine stimulation).